# WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Sunday, October 01, 2006

| Hide? | Set Name | Query | Hit Count |
|---|---|---|---|
| | | *DB=PGPB,USPT; PLUR=YES; OP=OR* | |
| ☐ | L24 | l17 and L23 | 36 |
| ☐ | L23 | (cluster or clustering) same (signature or data or profile) | 29683 |
| ☐ | L22 | l20 and l17 | 2 |
| ☐ | L21 | l20 and l17L20 | 0 |
| ☐ | L20 | (cluster or clustering) same signature | 990 |
| ☐ | L19 | (cluster or clustering) same signature and L17 | 2 |
| ☐ | L18 | (cluster or clustering) same (phenotype or phenotypic) same signature and L17 | 2 |
| ☐ | L17 | (invert or inverting) with (phase or $phase) | 11325 |
| ☐ | L16 | (invert or inverting) with (phase or $phase) and l7 | 5 |
| ☐ | L15 | (invert or inverting) with (phase or $phase) and l8 | 3 |
| ☐ | L14 | (invert or inverting) and l8 | 51 |
| ☐ | L13 | (invert or inversion or inverting) and l8 | 228 |
| ☐ | L12 | (invert or inversion or inverting) and l11 | 2 |
| ☐ | L11 | (cluster or clustering) same (phenotype or phenotypic) same signature same (library or screen or screening or microarray or array) | 8 |
| ☐ | L10 | l9 and (library or screen or screening or microarray or array) | 17 |
| ☐ | L9 | (cluster or clustering) same (phenotype or phenotypic) same signature | 17 |
| ☐ | L8 | (cluster or clustering) same (phenotype or phenotypic) | 1328 |
| ☐ | L7 | (cluster or clustering) and (phenotype or phenotypic) | 15836 |
| ☐ | L6 | (cluster or clustering) and L5 | 8 |
| ☐ | L5 | minor-james$.in. | 41 |
| ☐ | L4 | (cluster or clustering) and l1 | 1 |
| ☐ | L3 | (cluster or clustering) and l2 | 0 |
| ☐ | L2 | 5,860,917.pn. or 6,171,797.pn. or 6,180,351.pn. or 6,188,969.pn. or 6,222,664.pn. or 6,232,072.pn. or 6,242,266.pn. or 6,251,685.pn. or 6,320,196.pn. or 6,323,043.pn. or 6,355,921.pn. or 6,371,370.pn. or 6,406,849.pn. or 6,486,457.pn. or 6,518,556.pn. | 15 |
| ☐ | L1 | 20050037363.pn. | 1 |

END OF SEARCH HISTORY

Connecting via Winsock to STN

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LOGINID:SSSPTA1639MLS

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

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|---|---|---|
| NEWS 1 | | Web Page URLs for STN Seminar Schedule - N. America |
| NEWS 2 | | "Ask CAS" for self-help around the clock |
| NEWS 3 | FEB 27 | New STN AnaVist pricing effective March 1, 2006 |
| NEWS 4 | MAY 10 | CA/CAplus enhanced with 1900-1906 U.S. patent records |
| NEWS 5 | MAY 11 | KOREAPAT updates resume |
| NEWS 6 | MAY 19 | Derwent World Patents Index to be reloaded and enhanced |
| NEWS 7 | MAY 30 | IPC 8 Rolled-up Core codes added to CA/CAplus and USPATFULL/USPAT2 |
| NEWS 8 | MAY 30 | The F-Term thesaurus is now available in CA/CAplus |
| NEWS 9 | JUN 02 | The first reclassification of IPC codes now complete in INPADOC |
| NEWS 10 | JUN 26 | TULSA/TULSA2 reloaded and enhanced with new search and and display fields |
| NEWS 11 | JUN 28 | Price changes in full-text patent databases EPFULL and PCTFULL |
| NEWS 12 | JUl 11 | CHEMSAFE reloaded and enhanced |
| NEWS 13 | JUl 14 | FSTA enhanced with Japanese patents |
| NEWS 14 | JUl 19 | Coverage of Research Disclosure reinstated in DWPI |
| NEWS 15 | AUG 09 | INSPEC enhanced with 1898-1968 archive |
| NEWS 16 | AUG 28 | ADISCTI Reloaded and Enhanced |
| NEWS 17 | AUG 30 | CA(SM)/CAplus(SM) Austrian patent law changes |
| NEWS 18 | SEP 11 | CA/CAplus enhanced with more pre-1907 records |
| NEWS 19 | SEP 21 | CA/CAplus fields enhanced with simultaneous left and right truncation |
| NEWS 20 | SEP 25 | CA(SM)/CAplus(SM) display of CA Lexicon enhanced |
| NEWS 21 | SEP 25 | CAS REGISTRY(SM) no longer includes Concord 3D coordinates |
| NEWS 22 | SEP 25 | CAS REGISTRY(SM) updated with amino acid codes for pyrrolysine |
| NEWS 23 | SEP 28 | CEABA-VTB classification code fields reloaded with new classification scheme |

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|---|---|
| NEWS EXPRESS | JUNE 30 CURRENT WINDOWS VERSION IS V8.01b, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 26 JUNE 2006. |
| NEWS HOURS | STN Operating Hours Plus Help Desk Availability |
| NEWS LOGIN | Welcome Banner and News Items |
| NEWS IPC8 | For general information regarding STN implementation of IPC 8 |
| NEWS X25 | X.25 communication option no longer available |

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * * * * * * * * * * * * STN  Columbus  * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 15:56:56 ON 01 OCT 2006

=> e minor james/au
THIS COMMAND NOT AVAILABLE IN THE CURRENT FILE
Some commands only work in certain files.  For example, the EXPAND
command can only be used to look at the index in a file which has an
index.  Enter "HELP COMMANDS" at an arrow prompt (=>) for a list of
commands which can be used in this file.

=> fil medline biosis caplus scisearch embase wpids
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.42       0.42

FILE 'MEDLINE' ENTERED AT 15:57:55 ON 01 OCT 2006

FILE 'BIOSIS' ENTERED AT 15:57:55 ON 01 OCT 2006
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FILE 'CAPLUS' ENTERED AT 15:57:55 ON 01 OCT 2006
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FILE 'WPIDS' ENTERED AT 15:57:55 ON 01 OCT 2006
COPYRIGHT (C) 2006 THE THOMSON CORPORATION

=> e minor james/au
E1            3     MINOR J W/AU
E2           12     MINOR JACOB C/AU
E3            9 --> MINOR JAMES/AU
E4            3     MINOR JAMES B/AU
E5            3     MINOR JAMES C/AU
E6            3     MINOR JAMES CRAIG/AU
E7            1     MINOR JAMES E/AU
E8            1     MINOR JAMES G/AU
E9            2     MINOR JAMES I JR/AU
E10          17     MINOR JAMES L/AU
E11           1     MINOR JAMES LYELL/AU
E12          23     MINOR JAMES M/AU

=> e minor james m/au
E1           17     MINOR JAMES L/AU
E2            1     MINOR JAMES LYELL/AU
E3           23 --> MINOR JAMES M/AU
E4           10     MINOR JAMES R/AU
E5            1     MINOR JAN/AU
E6           14     MINOR JAN L/AU
E7            1     MINOR JENNIFER L/AU
E8            1     MINOR JERIS K/AU
E9            4     MINOR JESSE E/AU
E10           1     MINOR JESSIE/AU
E11          30     MINOR JESSIE E/AU
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E12          2     MINOR JOHN/AU

=> e3
L1          23 "MINOR JAMES M"/AU

=> (cluster or clustering) and l1
L2           3 (CLUSTER OR CLUSTERING) AND L1

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3           3 DUP REM L2 (0 DUPLICATES REMOVED)

=> d ibib abs l3 1-3

L3   ANSWER 1 OF 3  CAPLUS  COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER:         2005:1335580  CAPLUS
DOCUMENT NUMBER:          144:46258
TITLE:                    Treatment discovery based on comparative genomic
                          hybridization (CGH) analysis
INVENTOR(S):              Minor, James M.; Woo, Wilson
PATENT ASSIGNEE(S):       USA
SOURCE:                   U.S. Pat. Appl. Publ., 20 pp., Cont.-in-part of U.S.
                          Ser. No. 640,081.
                          CODEN: USXXCO
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   2
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.          DATE
     ---------------      ----   --------    --------------------     --------
     US 2005282227         A1    20051222    US 2005-215483           20050830
     US 2005037363         A1    20050217    US 2003-640081           20030813
PRIORITY APPLN. INFO.:                       US 2003-640081      A2 20030813
AB   Methods, systems and computer readable media for discovering a combination
     of treatments to reduce the progress of, or eliminate a tissue malady, are
     provided. The methods include the steps of: (a) measuring gene expression
     values of sample of tissue exhibiting the tissue malady and reference sample
     tissue that does not exhibit the malady, using CGH array designed to
     measure gene sequences and possible variations in gene sequences
     attributable to the malady; (b) generating gene expression signatures from
     differential expression values of ratios of the measured gene expression
     values between the sample exhibiting the malady and the reference sample,
     across all samples, resp.; (c) treating thetissue sample exhibiting the
     malady with a treatment; (d) measuring a treatment-response value with
     respect to each of the tissue samples treated, as effected by the
     treatment; (e) generating a phenotypic signature representing the
     treatment-response values of each of the tissue samples treated; (f)
     repeating steps (c)-(e) with a different treatment so that multiple
     phenotypic signatures have been generated for multiple treatments; (g)
     performing a clustering operation based on the gene expression
     signatures of the differential expression levels and the phenotypic
     signatures of the treatment-response values together; and (h) selecting
     treatments by identifying the treatment-response phenotypic signatures
     caused by those treatments. In addition, embodiments of the present
     invention further relate to computer readable media or computer program
     products that include program instructions and/or data (including data
     structures) for performing various computer-implemented operations. The
     present invention is a forward-looking way of choosing and predicting
     specific combinations of treatments to test, e.g., using high-throughput
     (HTP) screening of treatment combinations, and as such, greatly reduces
     the time to finding successful combinations, which currently have only
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been discovered accidentally, through hindsight and experiences gained through individual treatments. The treatments identified are targeted to the genes involved in the disease process/malady. Because of this, the chances of significant side effects are reduced. A technique for excluding potential treatments may also be carried out. One example of such an exclusion technique is to generate phenotypic signature representing treatment-response values of each of the tissue samples exhibiting the malady, resultant from treating the tissue samples exhibiting the malady, with treatment having known undesirable characteristics (e.g., is toxic to normal tissues, or ineffective, or has other undesirable side effects, etc.) for treatment of the tissues exhibiting the malady, using the techniques described above.

L3 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:1154196 CAPLUS
DOCUMENT NUMBER: 143:405367
TITLE: Methods and systems for differential clustering of gene expression profiles for high throughput identification of potential functionally variant genes
INVENTOR(S): Minor, James M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 18 pp.
CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005240357 | A1 | 20051027 | US 2004-831866 | 20040426 |
| PRIORITY APPLN. INFO.: | | | US 2004-831866 | 20040426 |

AB The present invention provides methods, systems and computer readable media for high throughput identification of potential functionally variant genes. Gene expression response profiles are generated for various sets of the samples and then differentially clustered across such sets to observe genes whose expression response profiles change cluster membership going from one set to another. Statistical anal. is performed with regard to the change from one cluster membership to another to determine whether the change from one cluster membership to another is statistically significant. If the change is determined to be statistically significant, the gene represented by the gene expression response profiles having been analyzed is identified as being a potential functionally variant gene. The nature of the function change may also be identified by the present systems, methods and computer readable media. The cluster emphasis is on the synchronization of profile trend variations rather than on shifts in expression levels. The present invention further covers forwarding a result, transmitting data representing a result and/or receiving a result obtained from any of the methods described herein.

L3 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:140656 CAPLUS
DOCUMENT NUMBER: 142:212296
TITLE: Methods, systems and recordable media for multi-drug treatment discovery using expression data characterizing protein pathways of diseases
INVENTOR(S): Minor, James M.
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 22 pp.
CODEN: USXXCO

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005037363 | A1 | 20050217 | US 2003-640081 | 20030813 |
| WO 2005017804 | A2 | 20050224 | WO 2004-US26366 | 20040813 |
| WO 2005017804 | A3 | 20060323 | | |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW

RW: AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG, BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

| | | | | |
|---|---|---|---|---|
| US 2005282227 | A1 | 20051222 | US 2005-215483 | 20050830 |

PRIORITY APPLN. INFO.: US 2003-640081 A 20030813

AB Methods, systems and recordable media are described for using expression data characterizing protein pathways of diseases to produce phase relationships between treatment responses of diseased tissues to treatments applied thereto and expression profiles of the diseased tissues as measured when untreated. Methods, systems and recordable media are also described for augmenting an original or existing treatment or treatment combination with one or more treatments that cover gene activity of a disease not addressed by the original/existing treatment. Application to screening treatments on lung cancer tissues are illustrated.

=> (cluster or clustering) and (phenotype or phenotypic) and signature and (library or screen or screening or microarray or array)
L4 59 (CLUSTER OR CLUSTERING) AND (PHENOTYPE OR PHENOTYPIC) AND SIGNATURE AND (LIBRARY OR SCREEN OR SCREENING OR MICROARRAY OR ARRAY)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 29 DUP REM L4 (30 DUPLICATES REMOVED)

=> (invert or inverting) and l5
L6 0 (INVERT OR INVERTING) AND L5

=> d ibib abs l5 1-29

L5 ANSWER 1 OF 29 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:388254 SCISEARCH
THE GENUINE ARTICLE: 029RD
TITLE: Gene expression profiling of breast cell lines identifies potential new basal markers
AUTHOR: Charafe-Jauffret E; Ginestier C; Monville F; Finetti P; Adelaide J; Cervera N; Fekairi S; Xerri L; Jacquemier J; Birnbaum D (Reprint); Bertucci F
CORPORATE SOURCE: Inst J Paoli I Calmettes, Marseille Canc Inst, Mol Oncol Lab, UMR599, Inserm, 27 Bd Lei Roure, F-13009 Marseille, France (Reprint); Inst J Paoli I Calmettes, Marseille Canc Inst, Mol Oncol Lab, UMR599, Inserm, F-13009 Marseille,

France; Inst J Paoli I Calmettes, Dept BioPathol, Marseille, France; Univ Mediterranee, UFR Med, Marseille, France; Inst Paoli Calmettes, INSERM, UMR599, Dept Med Oncol, Marseille Canc Inst, Marseille, France birnbaum@marseille.inserm.fr

COUNTRY OF AUTHOR: France

SOURCE: ONCOGENE, (APR 2006) Vol. 25, No. 15, pp. 2273-2284. ISSN: 0950-9232.

PUBLISHER: NATURE PUBLISHING GROUP, MACMILLAN BUILDING, 4 CRINAN ST, LONDON N1 9XW, ENGLAND.

DOCUMENT TYPE: Article; Journal

LANGUAGE: English

REFERENCE COUNT: 57

ENTRY DATE: Entered STN: 20 Apr 2006
Last Updated on STN: 20 Apr 2006

*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB A better molecular characterization of breast cell lines (BCL) may help discover new markers to apply to tumour samples. We performed gene and protein expression pro. ling of 31 BCL using whole-genome DNA microarrays and immunohistochemistry (IHC) on 'cell microarrays' (CMA), respectively. Global hierarchical clustering discriminated two groups of BCL: group I corresponded to luminal cell lines, group II to basal and mesenchymal cell lines. Correlations with centroids calculated from a published 'intrinsic 500-gene set' assigned 15 cell lines as luminal, eight as basal and four as mesenchymal. A set of 1.233 genes was differentially expressed between basal and luminal samples. Mesenchymal and basal subtypes were rather similar and discriminated by only 227 genes. The expression of 10 proteins (CAV1, CD44, EGFR, MET, ETS1, GATA3, luminal cytokeratin CK19, basal cytokeratin CK5/6, CD10, and ERM protein moesin) encoded by luminal vs basal discriminator genes confirmed the subtype classification and the validity of the identified markers. Our BCL basal/luminal signature correctly re-classified the published series of tumour samples that originally served to identify the molecular subtypes, suggesting that the identified markers should be useful for tumour classification and might represent promising targets for disease management.

L5 ANSWER 2 OF 29 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2006328183 MEDLINE

DOCUMENT NUMBER: PubMed ID: 16710476

TITLE: Met-regulated expression signature defines a subset of human hepatocellular carcinomas with poor prognosis and aggressive phenotype.

AUTHOR: Kaposi-Novak Pal; Lee Ju-Seog; Gomez-Quiroz Luis; Coulouarn Cedric; Factor Valentina M; Thorgeirsson Snorri S

CORPORATE SOURCE: Laboratory of Experimental Carcinogenesis, Center for Cancer Research, National Cancer Institute, NIH, Bethesda, Maryland 20892, USA.

SOURCE: The Journal of clinical investigation, (2006 Jun) Vol. 116, No. 6, pp. 1582-95. Electronic Publication: 2006-05-18. Journal code: 7802877. ISSN: 0021-9738.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200608

ENTRY DATE: Entered STN: 3 Jun 2006
Last Updated on STN: 2 Aug 2006
Entered Medline: 1 Aug 2006

AB Identification of specific gene expression signatures characteristic of oncogenic pathways is an important step toward molecular classification of human malignancies. Aberrant activation of the Met signaling pathway is

frequently associated with tumor progression and metastasis. In this study, we defined the Met-dependent gene expression signature using global gene expression profiling of WT and Met-deficient primary mouse hepatocytes. Newly identified transcriptional targets of the Met pathway included genes involved in the regulation of oxidative stress responses as well as cell motility, cytoskeletal organization, and angiogenesis. To assess the importance of a Met-regulated gene expression signature, a comparative functional genomic approach was applied to 242 human hepatocellular carcinomas (HCCs) and 7 metastatic liver lesions. Cluster analysis revealed that a subset of human HCCs and all liver metastases shared the Met-induced expression signature. Furthermore, the presence of the Met signature showed significant correlation with increased vascular invasion rate and microvessel density as well as with decreased mean survival time of HCC patients. We conclude that the genetically defined gene expression signatures in combination with comparative functional genomics constitute an attractive paradigm for defining both the function of oncogenic pathways and the clinically relevant subgroups of human cancers.

L5 ANSWER 3 OF 29 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2006033834 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16273092
TITLE: Oncogenic pathway signatures in human cancers as a guide to targeted therapies.
AUTHOR: Bild Andrea H; Yao Guang; Chang Jeffrey T; Wang Quanli; Potti Anil; Chasse Dawn; Joshi Mary-Beth; Harpole David; Lancaster Johnathan M; Berchuck Andrew; Olson John A Jr; Marks Jeffrey R; Dressman Holly K; West Mike; Nevins Joseph R
CORPORATE SOURCE: Institute for Genome Sciences and Policy, Duke University, Durham, North Carolina 27708, USA.
SOURCE: Nature, (2006 Jan 19) Vol. 439, No. 7074, pp. 353-7. Electronic Publication: 2005-11-06. Journal code: 0410462. E-ISSN: 1476-4687.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 20 Jan 2006
Last Updated on STN: 15 Feb 2006
Entered Medline: 14 Feb 2006

AB The development of an oncogenic state is a complex process involving the accumulation of multiple independent mutations that lead to deregulation of cell signalling pathways central to the control of cell growth and cell fate. The ability to define cancer subtypes, recurrence of disease and response to specific therapies using DNA microarray-based gene expression signatures has been demonstrated in multiple studies. Various studies have also demonstrated the potential for using gene expression profiles for the analysis of oncogenic pathways. Here we show that gene expression signatures can be identified that reflect the activation status of several oncogenic pathways. When evaluated in several large collections of human cancers, these gene expression signatures identify patterns of pathway deregulation in tumours and clinically relevant associations with disease outcomes. Combining signature-based predictions across several pathways identifies coordinated patterns of pathway deregulation that distinguish between specific cancers and tumour subtypes. Clustering tumours based on pathway signatures further defines prognosis in respective patient subsets, demonstrating that patterns of oncogenic pathway deregulation underlie the development of the oncogenic phenotype and reflect the biology and outcome of specific cancers. Predictions of pathway deregulation in cancer cell

lines are also shown to predict the sensitivity to therapeutic agents that target components of the pathway. Linking pathway deregulation with sensitivity to therapeutics that target components of the pathway provides an opportunity to make use of these oncogenic pathway signatures to guide the use of targeted therapeutics.

L5 ANSWER 4 OF 29 MEDLINE on STN
ACCESSION NUMBER: 2006065688 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16100004
TITLE: Unique gene expression signature by human embryonic stem cells cultured under serum-free conditions correlates with their enhanced and prolonged growth in an undifferentiated stage.
AUTHOR: Skottman Heli; Stromberg Anne-Marie; Matilainen Eija; Inzunza Jose; Hovatta Outi; Lahesmaa Riitta
CORPORATE SOURCE: Turku Centre for Biotechnology, University of Turku, and REGEA Institute for Regenerative Medicine, Tampere University Hospital, 33520 Tampere, Finland.. Heli.Skottman@regea.fi
SOURCE: Stem cells (Dayton, Ohio), (2006 Jan) Vol. 24, No. 1, pp. 151-67. Electronic Publication: 2005-08-11. Journal code: 9304532. ISSN: 1066-5099.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200606
ENTRY DATE: Entered STN: 2 Feb 2006
Last Updated on STN: 10 Jun 2006
Entered Medline: 9 Jun 2006

AB Understanding the interaction between human embryonic stem cells (hESCs) and their microenvironment is crucial for the propagation and the differentiation of hESCs for therapeutic applications. hESCs maintain their characteristics both in serum-containing and serum-replacement (SR) media. In this study, the effects of the serum-containing and SR culture media on the gene expression profiles of hESCs were examined. Although the expression of many known embryonic stem cell markers was similar in cells cultured in either media, surprisingly, 1,417 genes were found to be differentially expressed when hESCs cultured in serum-containing medium were compared with those cultured in SR medium. Several genes upregulated in cells cultured in SR medium suggested increased metabolism and proliferation rates in this medium, providing a possible explanation for the increased growth rate of nondifferentiated cells observed in SR culture conditions compared with that in serum medium. Several genes characteristic for cells with differentiated phenotype were expressed in cells cultured in serum-containing medium. Our data clearly indicate that the manipulation of hESC culture conditions causes phenotypic changes of the cells that were reflected also at the level of gene expression. Such changes may have fundamental importance for hESCs, and gene expression changes should be monitored as a part of cell culture optimization aiming at a clinical use of hESCs for cell transplantation.

L5 ANSWER 5 OF 29 CAPLUS COPYRIGHT 2006 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2005:1335580 CAPLUS
DOCUMENT NUMBER: 144:46258
TITLE: Treatment discovery based on comparative genomic hybridization (CGH) analysis
INVENTOR(S): Minor, James M.; Woo, Wilson
PATENT ASSIGNEE(S): USA
SOURCE: U.S. Pat. Appl. Publ., 20 pp., Cont.-in-part of U.S. Ser. No. 640,081.

CODEN: USXXCO
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| US 2005282227 | A1 | 20051222 | US 2005-215483 | 20050830 |
| US 2005037363 | A1 | 20050217 | US 2003-640081 | 20030813 |

PRIORITY APPLN. INFO.: US 2003-640081 A2 20030813

AB Methods, systems and computer readable media for discovering a combination of treatments to reduce the progress of, or eliminate a tissue malady, are provided. The methods include the steps of: (a) measuring gene expression values of sample of tissue exhibiting the tissue malady and reference sample tissue that does not exhibit the malady, using CGH array designed to measure gene sequences and possible variations in gene sequences attributable to the malady; (b) generating gene expression signatures from differential expression values of ratios of the measured gene expression values between the sample exhibiting the malady and the reference sample, across all samples, resp.; (c) treating thetissue sample exhibiting the malady with a treatment; (d) measuring a treatment-response value with respect to each of the tissue samples treated, as effected by the treatment; (e) generating a phenotypic signature representing the treatment-response values of each of the tissue samples treated; (f) repeating steps (c)-(e) with a different treatment so that multiple phenotypic signatures have been generated for multiple treatments; (g) performing a clustering operation based on the gene expression signatures of the differential expression levels and the phenotypic signatures of the treatment-response values together; and (h) selecting treatments by identifying the treatment-response phenotypic signatures caused by those treatments. In addition, embodiments of the present invention further relate to computer readable media or computer program products that include program instructions and/or data (including data structures) for performing various computer-implemented operations. The present invention is a forward-looking way of choosing and predicting specific combinations of treatments to test, e.g., using high-throughput (HTP) screening of treatment combinations, and as such, greatly reduces the time to finding successful combinations, which currently have only been discovered accidentally, through hindsight and experiences gained through individual treatments. The treatments identified are targeted to the genes involved in the disease process/malady. Because of this, the chances of significant side effects are reduced. A technique for excluding potential treatments may also be carried out. One example of such an exclusion technique is to generate phenotypic signature representing treatment-response values of each of the tissue samples exhibiting the malady, resultant from treating the tissue samples exhibiting the malady, with treatment having known undesirable characteristics (e.g., is toxic to normal tissues, or ineffective, or has other undesirable side effects, etc.) for treatment of the tissues exhibiting the malady, using the techniques described above.

L5 ANSWER 6 OF 29 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2005-172243 [18] WPIDS
CROSS REFERENCE: 2006-055107 [06]
DOC. NO. NON-CPI: N2005-143762
DOC. NO. CPI: C2005-055243
TITLE: Screening multiple treatments for complex diseases, e.g. cancer, by treating diseased tissue samples, measuring treatment-response values, generating phenotypic signatures, performing

clustering operation, and selecting treatment.
DERWENT CLASS: B04 B05 D16 S03 T01
INVENTOR(S): MINOR, J; MINOR, J M
PATENT ASSIGNEE(S): (MINO-I) MINOR J M; (AGIL-N) AGILENT TECHNOLOGIES INC
COUNTRY COUNT: 109
PATENT INFORMATION:

| PATENT NO | KIND | DATE | WEEK | LA | PG |
|---|---|---|---|---|---|
| US 2005037363 | A1 | 20050217 | (200518)* | | 22 |
| WO 2005017804 | A2 | 20050224 | (200518) | EN | |
| RW: | AT BE BG BW CH CY CZ DE DK EA EE ES FI FR GB GH GM GR HU IE IT KE LS LU MC MW MZ NA NL OA PL PT RO SD SE SI SK SL SZ TR TZ UG ZM ZW | | | | |
| W: | AE AG AL AM AT AU AZ BA BB BG BR BW BY BZ CA CH CN CO CR CU CZ DE DK DM DZ EC EE EG ES FI GB GD GE GH GM HR HU ID IL IN IS JP KE KG KP KR KZ LC LK LR LS LT LU LV MA MD MG MK MN MW MX MZ NA NI NO NZ OM PG PH PL PT RO RU SC SD SE SG SK SL SY TJ TM TN TR TT TZ UA UG US UZ VC VN YU ZA ZM ZW | | | | |
| EP 1654686 | A2 | 20060510 | (200632) | EN | |
| R: | AL AT BE BG CH CY CZ DE DK EE ES FI FR GB GR HR HU IE IT LI LT LU LV MC MK NL PL PT RO SE SI SK TR | | | | |

APPLICATION DETAILS:

| PATENT NO | KIND | APPLICATION | DATE |
|---|---|---|---|
| US 2005037363 | A1 | US 2003-640081 | 20030813 |
| WO 2005017804 | A2 | WO 2004-US26366 | 20040813 |
| EP 1654686 | A2 | EP 2004-781107 | 20040813 |
| | | WO 2004-US26366 | 20040813 |

FILING DETAILS:

| PATENT NO | KIND | PATENT NO |
|---|---|---|
| EP 1654686 | A2 Based on | WO 2005017804 |

PRIORITY APPLN. INFO: US 2003-640081 20030813
AN 2005-172243 [18] WPIDS
CR 2006-055107 [06]
AB US2005037363 A UPAB: 20060518
NOVELTY - Screening of a combination of treatments to specifically target a disease process comprises providing differential expression levels of diseased tissue samples relative to reference tissue, providing a phenotypic/ genotypic signature representing differential expression level for each tissue sample, treating diseased tissue samples with different treatments, measuring treatment-response values, generating phenotypic signatures, performing a clustering operation, and selecting treatments.

DETAILED DESCRIPTION - Screening of a combination of treatments to specifically target a disease process comprises providing differential expression levels of diseased tissue samples relative to reference tissue for features of microarrays used to calculate the differential expression levels; for respective features of microarrays for each diseased tissue sample, providing a phenotypic/ genotypic signature representing the differential expression level for each tissue sample for that feature, respectively; treating the diseased tissue samples with a treatment; measuring a treatment-response value with respect to each of the diseased tissue samples as effected by the treatment; generating a phenotypic signature representing the treatment-response values of each of the diseased tissue samples; performing different treatment(s) so that multiple

phenotypic signatures have been generated for multiple treatments; performing a clustering operation based on the phenotypic/genotypic signatures of the differential expression levels and the phenotypic signatures of the treatment-response values together; and selecting treatments by identifying the treatment-response phenotypic signatures caused by those treatments, and which are clustered with phenotypic signatures representing differential expression levels representative of the diseased tissue samples.

INDEPENDENT CLAIMS are also included for:

(1) a method comprising forwarding a result obtained from the inventive screening method to a remote location;

(2) a method comprising transmitting data representing a result obtained from the inventive screening method to a remote location;

(3) a method comprising receiving a result obtained from the inventive screening method;

(4) a method of augmenting an original or existing single treatment or treatment combination for a disease with additional treatment that covers gene activity of the disease not addressed by the original or existing treatment;

(5) a system for screening a combination of treatments to specifically target a disease process;

(6) a method for determining phase relationships between treatment responses of diseased tissues to treatments that are applied and expression profiles of the diseased tissues; and

(7) a computer readable medium carrying sequences of instructions for the inventive screening method.

USE - For screening and finding multiple treatments for complex diseases, such as cancers, viral diseases, e.g. AIDS and SARS, and drug-resistant bacteria, e.g. tuberculosis.

ADVANTAGE - The method can be used more effectively than a single treatment approach to treating disease.

Dwg.0/8

L5 ANSWER 7 OF 29 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 2005365987 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16024604
TITLE: The RhoGAP protein DLC-1 functions as a metastasis suppressor in breast cancer cells.
AUTHOR: Goodison Steve; Yuan Jing; Sloan Derek; Kim Ryung; Li Cheng; Popescu Nicholas C; Urquidi Virginia
CORPORATE SOURCE: Department of Pathology, University of Florida Health Science Center, Shands Hospital, Jacksonville, Florida 32209-6511, USA.. steve.goodison@jax.ufl.edu
CONTRACT NUMBER: CA R01 108597 (NCI)
R01 CA108597-01 (NCI)
SOURCE: Cancer research, (2005 Jul 15) Vol. 65, No. 14, pp. 6042-53.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200509
ENTRY DATE: Entered STN: 19 Jul 2005
Last Updated on STN: 15 Sep 2005
Entered Medline: 14 Sep 2005

AB The identification of molecular signatures characteristic of tumor cells that are capable of metastatic spread is required for the development of therapeutic interventions to abrogate this lethal process. To facilitate this, we have previously characterized an experimental system in which the

role of candidate metastasis-related genes can be screened and tested. Monoclonal cell lines M4A4 and NM2C5 are spontaneously occurring sublines of the MDA-MB-435 cell breast tumor cell line that exhibit phenotypic differences in growth, invasion, and metastatic efficiency in athymic mice. In this study, transcriptional profiles of these cell lines were created using oligonucleotide microarrays representing over 12,000 genes. Intensity modeling and hierarchical clustering analysis identified a 171-gene expression signature that correlated with metastatic phenotype and highlighted several GTPase signaling components. Restoration of one of these GTPases, deleted in liver cancer-1 (DLC-1), in metastatic M4A4 cells to levels observed in the nonmetastatic NM2C5 cell line resulted in the inhibition of migration and invasion in vitro and a significant reduction in the ability of these cells to form pulmonary metastases in athymic mice. These studies show the utility of expression profiling, in an appropriate experimental system, to identify genetic determinants of metastatic sufficiency. The finding that DLC-1 can act as a metastasis-suppressor gene supports an influential role for GTPase signaling in tumor progression.

L5 ANSWER 8 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:243573 BIOSIS
DOCUMENT NUMBER: PREV200510033215
TITLE: A SUMO ligase is part of a nuclear multiprotein complex that affects DNA repair and chromosomal organization.
AUTHOR(S): Zhao, Xiaolan; Blobel, Gunter [Reprint Author]
CORPORATE SOURCE: Rockefeller Univ, Howard Hughes Med Inst, Cell Biol Lab, 1230 York Ave, New York, NY 10021 USA
blobel@rockefeller.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (MAR 29 2005) Vol. 102, No. 13, pp. 4777-4782.
CODEN: PNASA6. ISSN: 0027-8424.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 29 Jun 2005
Last Updated on STN: 29 Jun 2005

AB Through a genetic screen using myosin-like protein strains mlp1 Delta mlp2 Delta and biochemical purification, we identified a complex of eight proteins, each required for growth and DNA repair in Saccharomyces cerevisiae. Among the subunits are Mms21 that contains a putative Siz/PIAS (protein inhibitor of activated signal transducer and activator of transcription) RING domain characteristic of small ubiquitin-like modifier(SUMO) ligases, two structural-maintenance-of-chromosome (Smc) proteins, Smc5 and Smc6, and a protein that contains an ubiquitin ligase signature domain. We show that these proteins colocalized to several distinct nuclear foci. Biochemical and genetic data demonstrated that Mms21 indeed functions as a SUIMO ligase and that this activity requires the Siz/PIAS (protein inhibitor of activated signal transducer and activator of transcription) RING domain. The substrates for this SUMO ligase include a subunit of the octameric complex, Smc5, and the DNA repair protein Yku70. We further show that the abolition of the SUMO E3 activity of Mms21 leads to such disparate phenotypes as DNA damage sensitivity, defects in nucleolar integrity and telomere clustering, silencing, and length regulation. We propose that Mms21 sumoylates proteins involved in these diverse processes and that the other members of the complex, particularly Smc5/6, facilitate proper substrate sumoylation by localizing Mms21 to specific chromosomal regions.

L5 ANSWER 9 OF 29 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN
ACCESSION NUMBER: 2005345954 EMBASE

TITLE: Conservation of unique cell-surface CD antigen mosaics in HIV-1-infected individuals.
AUTHOR: Woolfson A.; Stebbing J.; Tom B.D.M.; Stoner K.J.; Gilks W.R.; Kreil D.P.; Mulligan S.P.; Belov L.; Chrisp J.S.; Errington W.; Wildfire A.; Erber W.N.; Bower M.; Gazzard B.; Christopherson R.I.; Scott M.A.
CORPORATE SOURCE: A. Woolfson, University of Cambridge School of Clinical Medicine, Addenbrooke's Hospital, Box 111, Hills Road, Cambridge CB2 2SP, United Kingdom. awl35@cam.ac.uk
SOURCE: Blood, (1 Aug 2005) Vol. 106, No. 3, pp. 1003-1007. .
Refs: 32
ISSN: 0006-4971 CODEN: BLOOAW
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
006 Internal Medicine
025 Hematology
026 Immunology, Serology and Transplantation
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English
ENTRY DATE: Entered STN: 1 Sep 2005
Last Updated on STN: 1 Sep 2005

AB Cluster of differentiation (CD) antigens are expressed on cells of myeloid and lymphoid lineages. As most disease processes involve immune system activation or suppression, these antigens offer unique opportunities for monitoring host responses. Immunophenotyping using limited numbers of CD antigens enables differentiation states of immune system cells to be determined. Extended phenotyping involving parallel measurement of multiple CD antigens may help identify expression pattern signatures associated with specific disease states. To explore this possibility we have made a CD monoclonal antibody array and scanner, enabling the parallel immunophenotyping of leukocyte cell suspensions in a single and rapid analysis. To demonstrate this approach, we used the specific example of patients infected with human immunodeficiency virus type-1 (HIV-1). An invariant HIV-induced CD antigen signature has been defined that is both robust and independent of clinical outcome, composed of a unique profile of CD antigen expression levels that are both increased and decreased relative to internal controls. The results indicate that HIV-induced changes in CD antigen expression are disease specific and independent of outcome. Their invariant nature indicates an irreversible component to retroviral infection and suggests the utility of CD antigen expression patterns in other disease settings. .COPYRGT. 2005 by The American Society of Hematology.

L5 ANSWER 10 OF 29 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:700485 CAPLUS
DOCUMENT NUMBER: 143:283609
TITLE: Acute myeloid leukemia bearing cytoplasmic nucleophosmin (NPMc+ AML) shows a distinct gene expression profile characterized by up-regulation of genes involved in stem-cell maintenance
AUTHOR(S): Alcalay, Myriam; Tiacci, Enrico; Bergomas, Roberta; Bigerna, Barbara; Venturini, Elisa; Minardi, Simone P.; Meani, Natalia; Diverio, Daniela; Bernard, Loris; Tizzoni, Laura; Volorio, Sara; Luzi, Lucilla; Colombo, Emanuela; Lo Coco, Francesco; Meucci, Cristina; Falini, Brunangelo; Pelicci, Pier Giuseppe
CORPORATE SOURCE: The Gruppo Italiano Malattie Ematologiche Maligne dell'Adulto GIMEMA Acute Leukemia Working Party, Institute of Molecular Oncology (IFOM) of the Italian

Foundation for Cancer Research, Milan, Italy
SOURCE: Blood (2005), 106(3), 899-902
CODEN: BLOOAW; ISSN: 0006-4971
PUBLISHER: American Society of Hematology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Approx. one third of acute myeloid leukemias (AMLs) are characterized by aberrant cytoplasmic localization of nucleophosmin (NPMc+ AML), consequent to mutations in the NPM putative nucleolar localization signal. These events are mutually exclusive with the major AML-associated chromosomal rearrangements, and are frequently associated with normal karyotype, FLT3 mutations, and multilineage involvement. We report the gene expression profiles of 78 de novo AMLs (72 with normal karyotype; 6 without major chromosomal abnormalities) that were characterized for the subcellular localization and mutation status of NPM. Unsupervised clustering clearly separated NPMc+ from NPMc- AMLs, regardless of the presence of FLT3 mutations or non-major chromosomal rearrangements, supporting the concept that NPMc+ AML represents a distinct entity. The mol. signature of NPMc+ AML includes up-regulation of several genes putatively involved in the maintenance of a stem-cell phenotype, suggesting that NPMc+ AML may derive from a multipotent hematopoietic progenitor.

REFERENCE COUNT: 27 THERE ARE 27 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 11 OF 29 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2005:319752 SCISEARCH
THE GENUINE ARTICLE: 905TE
TITLE: Skeletal muscle gene expression profiling in mitochondrial disorders
AUTHOR: Crimi M (Reprint); Bordoni A; Menozzi G; Riva L; Fortunato F; Galbiati S; Del Bo R; Pozzoli U; Bresolin N; Comi G P
CORPORATE SOURCE: Univ Milan, Dept Neurol Sci, Pad Ne Ponti, Via F Sforza 35, I-20122 Milan, Italy (Reprint); Univ Milan, Dept Neurol Sci, I-20122 Milan, Italy; IRCCS, Osped Maggiore Policlin, Dino Ferrari Ctr, I-20122 Milan, Italy; CEND, I-20122 Milan, Italy; IRCCS E Medea La Nostra Famiglia, I-23842 Bosisio Parini, LC, Italy; Polytech Univ, Dept Biomed Engn, I-20133 Milan, Italy
marcreamy@tiscali.it
COUNTRY OF AUTHOR: Italy
SOURCE: FASEB JOURNAL, (FEB 2005) Vol. 19, No. 2, pp. 866-+.
ISSN: 0892-6638.
PUBLISHER: FEDERATION AMER SOC EXP BIOL, 9650 ROCKVILLE PIKE, BETHESDA, MD 20814-3998 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 55
ENTRY DATE: Entered STN: 31 Mar 2005
Last Updated on STN: 28 Sep 2006

*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Extremely variable clinic and genetic features characterize mitochondrial encephalomyopathy (ME M). Pathogenic mitochondrial DNA(mtDNA) defects can be divided into large-scale rearrangements and single point mutations. Clinical manifestations become evident when a threshold percentage of the total mtDNA is mutated. In some MEM, the "mutant load" in an affected tissue is directly related to the severity of the phenotype. However, the clinical phenotype is not simply a direct consequence of the relative abundance of mutated mtDNA. Other factors, such as nuclear background, can contribute to the disease process, resulting in a wide range of phenotypes caused by the same mutation. Using Affymetrix oligonucleotide cDNA microarrays(HG-U133A), we

studied the gene expression profile of muscle tissue biopsies obtained from 12 MEM patients[4 common 4977 bp deleted mtDNA and 8 A3243G:4 progressive external ophthalmoplegia (PEO) and 4 mitochondrial myopathy,encephalopathy, lactic cidosis, and stroke-like episodes syndrome (MELAS) phenotypes] compared with age matched healthy individuals. We found several differentially expressed genes: 35 were markedly up-regulated in the mtDNA macro-deletion group(vs. the control group) and 4 decreased; 56 genes were dysregulated in A3243G-related disorders(53 down-regulated in PEO and 3 upregulated in MELAS). Finally, 12 genes were similarly regulated in the majority of the MEM patients under study. Amongst these, we identified an increased expression of genes related to the metabolism of the amino groups, as well as of several genes involved in genetic information processing. Moreover, few genes were similarly decreased in MEM patients vs. the control group. Real-time PCR demonstrated excellent reproducibility of the microarray-based findings. The observed expression changes are likely to represent a molecular signature for mitochondrial disorders. Furthermore,the differential expression profile of MELAS(A3243G) vs. PEOA3243G may support a role of nuclear background in contributing to these different clinical phenotypes. MEM microarray data are available from GEO database (http://www.ncbi.nlm.nih.gov/geo/) wit h the access ion number: GSE1462.

L5 ANSWER 12 OF 29 CAPLUS COPYRIGHT 2006 ACS on STN
ACCESSION NUMBER: 2005:385940 CAPLUS
DOCUMENT NUMBER: 143:319998
TITLE: Systematic deletion analysis of fission yeast protein kinases
AUTHOR(S): Bimbo, Andrea; Jia, Yonghui; Poh, Siew Lay; Karuturi, R. Krishna Murthy; den Elzen, Nicole; Peng, Xu; Zheng, Liling; O'Connell, Matthew; Liu, Edison T.; Balasubramanian, Mohan K.; Liu, Jianhua
CORPORATE SOURCE: Temasek Life Sciences Laboratory, 1 Research Link, NUS, Singapore, 117604, Singapore
SOURCE: Eukaryotic Cell (2005), 4(4), 799-813
CODEN: ECUEA2; ISSN: 1535-9778
PUBLISHER: American Society for Microbiology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Eukaryotic protein kinases are key mols. mediating signal transduction that play a pivotal role in the regulation of various biol. processes, including cell cycle progression, cellular morphogenesis, development, and cellular response to environmental changes. A total of 106 eukaryotic protein kinase catalytic domain-containing proteins have been found in the entire fission yeast genome, 44% (or 64%) of which possess orthologs (or nearest homologs) in humans, based on sequence similarity within catalytic domains. Systematic deletion anal. of all putative protein kinase-encoding genes have revealed that 17 out of 106 were essential for viability, including three previously uncharacterized putative protein kinases. Although the remaining 89 protein kinase mutants were able to form colonies under optimal growth conditions, 46% of the mutants exhibited hypersensitivity to at least 1 of the 17 different stress factors tested. Phenotypic assessment of these mutants allowed us to arrange kinases into functional groups. Based on the results of this assay, we propose also the existence of four major signaling pathways that are involved in the response to 17 stresses tested. Microarray anal. demonstrated a significant correlation between the expression signature and growth phenotype of kinase mutants tested. Our complete microarray data sets are available at http://giscompute.gis.a-star.edu.sg/.apprx.gisljh/kinome.
REFERENCE COUNT: 83 THERE ARE 83 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2006:181476 BIOSIS
DOCUMENT NUMBER: PREV200600183588
TITLE: Gene expression profiling of bulk CD138+plasma-cells in preneoplastic gammopathy/asymptomatic myeloma identifies distinct subsets of patients.
AUTHOR(S): Dhodapkar, Madhav V. [Reprint Author]; Zhan, Fenghuang; Rasmussen, Erik; Burington, Bart; Durie, Brian; Crowley, John; Barlogie, Bart; Shaughnessy, John
CORPORATE SOURCE: Rockefeller Univ, Lab Tumor Immunol and Immunotherapy, New York, NY 10021 USA
SOURCE: Blood, (NOV 16 2005) Vol. 106, No. 11, Part 1, pp. 355A. Meeting Info.: 47th Annual Meeting of the American-Society-of-Hematology. Atlanta, GA, USA. December 10 -13, 2005. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Mar 2006
Last Updated on STN: 15 Mar 2006

AB Gene expression profiling of plasma cells (GEP-PC) has provided major insights into myeloma pathobiology. However the data about GEP-PC in preneoplastic gammopathy (MGUS) or asymptomatic myeloma (AM) are limited, and gene expression patterns that might predict outcome in these patients have not been defined. We analyzed GEP (using U133PIus Affymetrix microarrays), of plasma cells isolated by immuno-magnetic bead selection with CD138 microbeads, from the bone marrow of patients with MGUS (n = 16) and asymptomatic myeloma (AM; n = 18) enrolled in a prospective South West Oncology Group (SWOG) observational study. Data from normal plasma cells (PCs) and from 105 myeloma PCs were included as controls. Myeloma PCs were randomly selected to include at least 15 patients from each of the 7 subgroups previously identified based on GEP of myeloma tumor cells (Zhan and Shaughnessy, ASH 2004). After the suppression of immunoglobulin (Ig) genes, there were 1297 genes that significantly differed in expression between MGUS-PCs and MM-PCs, and 1099 genes that differed between MGUS-PCs and normal PCs with a 1% false discovery rate. Hierarchical cluster analysis of all samples was performed using 1000 plasma cell signature genes that were most differentially expressed between normal and myeloma PCs. These data demonstrated that both MGUS and AM samples were distributed between normal and MM samples. A prediction analysis of microarrays (PAM) model (PNAS 99:6567, 2002) utilizing 134 genes was then developed to determine if the signature from these genes in MGUS/AM was more similar to normal or to myeloma plasma cells. In this analysis, 11/16 (69%) of the MGUS samples were more similar to normal PC, compared to 6/18 (33%) of the AM samples (p = 0.04). At present, there are no reliable phenotypic markers to distinguish between normal and malignant PCs within the bulk CD 138+ population. Gene expression spikes for cyclin D1 and MAF/MAF-B were seen in both MGUS and AM cohorts, including in some patients with normal PC signature. These data provide the largest comparison to date, of GEP of PCs in preneoplastic versus malignant gammopathies and suggest that GEP may be a useful tool to prospectively identify subsets of patients within the MGUS/AM population with dominant normal PC or MM PC signatures, and potentially differing prognosis. Further analysis of differentially expressed genes between MGUS/MM PCs identified in this dataset may allow insights into the genomic changes in tumor cells underlying the malignant progression of myeloma.

L5 ANSWER 14 OF 29 MEDLINE on STN DUPLICATE 5

ACCESSION NUMBER: 2005549715 MEDLINE
DOCUMENT NUMBER: PubMed ID: 16172796
TITLE: Distinct molecular signature of inflammatory breast cancer by cDNA microarray analysis.
AUTHOR: Van Laere Steven; Van der Auwera Ilse; Van den Eynden Gert G; Fox Stephen B; Bianchi Fabrizio; Harris Adrian L; van Dam Peter; Van Marck Eric A; Vermeulen Peter B; Dirix Luc Y
CORPORATE SOURCE: Translational Cancer Research Group, Lab Pathology, University of Antwerp/University Hospital Antwerp, Edegem and General Hospital, Sint-Augustinus, Wilrijk, Belgium.
SOURCE: Breast cancer research and treatment, (2005 Oct) Vol. 93, No. 3, pp. 237-46.
Journal code: 8111104. ISSN: 0167-6806.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200512
ENTRY DATE: Entered STN: 18 Oct 2005
Last Updated on STN: 28 Dec 2005
Entered Medline: 27 Dec 2005

AB Inflammatory breast cancer (IBC) is a clinically distinct and aggressive form of locally advanced breast cancer with largely unknown genetic determinants. Overexpression of the RhoC GTPase and of HER2, and decreased ER-expression are involved in IBC. Multimodality treatment has increased survival but prognosis is still poor. Novel molecular targets for improved neoadjuvant treatment are necessary. Using cDNA microarrays, we performed genome-wide expression profiling of pre-treatment tumour samples of 16 patients with IBC and 18 patients with non-stage-matched non-IBC. Rigid clinical diagnostic criteria according to the TNM classification of the American Joint Committee on Cancer were adopted. Unsupervised hierarchical clustering accurately distinguished IBC and non-IBC samples. A set of 50 discriminator genes was identified in a learning group of tumour samples and was successful in diagnosing IBC in a validation group of samples (accuracy of 88%). Exclusion of ER-related or HER2-related genes did not alter this discriminatory accuracy, indicating that the expression of other genes in addition to ER and HER2 characterize the IBC phenotype. The molecular signature of IBC revealed the overexpression of a large number of NF-kappaB target genes, explaining at least part of the aggressive nature of IBC. Successful validation of some of the overexpressed genes by immunohistochemistry or real-time quantitative PCR demonstrated the robustness of the cDNA microarray experiments. The results of our study provide potential targets for the treatment of patients with IBC.

L5 ANSWER 15 OF 29 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2005:96674 SCISEARCH
THE GENUINE ARTICLE: 886YQ
TITLE: Transcriptional profiling and assessment of cell lines as in vitro models for mantle cell lymphoma
AUTHOR: Ek S; Ortega E; Borrebaeck C A K (Reprint)
CORPORATE SOURCE: Lund Univ, Dept Immunotechnol, POB 7031, SE-22007 Lund, Sweden (Reprint); Lund Univ, Dept Immunotechnol, SE-22007 Lund, Sweden
sara.ek@ummun.lth.se; carl.borrebaeck@immun.lth.se
COUNTRY OF AUTHOR: Sweden
SOURCE: LEUKEMIA RESEARCH, (FEB 2005) Vol. 29, No. 2, pp. 205-213.
ISSN: 0145-2126.
PUBLISHER: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD OX5 1GB, ENGLAND.

DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 35
ENTRY DATE: Entered STN: 3 Feb 2005
Last Updated on STN: 3 Feb 2005
*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Mantle cell lymphoma (MCL) is an aggressive malignancy and new treatment modalities must be established to increase patient survival time. In the search for new therapeutic targets, reliable and well-characterised in vitro models are essential. In this study, we have characterised three MCL cell lines (SP53, Granta 519 and NCEB1) in comparison with primary tumors front MCL, follicular lymphomas (FL), a(.)FL cell line(RL), a Burkitt lymphoma cell line (RAJI) and five different B cell populations from healthy individuals. Expression profiling was used to determine the relative expression of >12000 transcripts in these samples, and flow cytometry analysis was performed to establish a phenotypic signature for each of the cell lines. In addition, the cell lines were sequenced, and the frequency of somatic Mutations and immunoglobulin (Ig) variable heavy chain (V-H) Usage were determined.

We show by hierarchical clustering that the cell lines retain a genetic signature similar to primary MCL, which readily separated the MCL samples front the other lymphoma cell lines and the FL tumours. Furthermore, the MCL cell lines showed differences in the frequency of V-H somatic mutations (0-2.1%). The increased number of mutations in NCEB1, compared to the other MCL cell lines, was in agreement with a decreased expression of CD31, CD44, CXCR5, CCR7 and CCR6. Taken together, our data show that the cell lines are clearly derived from MCL tumours and expressed similar genetic and phenotypic signatures compared to primary tumours, which confirmed their usefulness as in vitro models. 

L5 ANSWER 16 OF 29 MEDLINE on STN
ACCESSION NUMBER: 2005329143 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15868446
TITLE: BRCA1 promoter methylation in sporadic breast tumors: relationship to gene expression profiles.
AUTHOR: Matros Evan; Wang Zhigang C; Lodeiro Gabriela; Miron Alexander; Iglehart J Dirk; Richardson Andrea L
CORPORATE SOURCE: Department of Surgery, Brigham and Women's Hospital, Boston, MA 02115, USA.
SOURCE: Breast cancer research and treatment, (2005 May) Vol. 91, No. 2, pp. 179-86.
Journal code: 8111104. ISSN: 0167-6806.
PUB. COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200507
ENTRY DATE: Entered STN: 29 Jun 2005
Last Updated on STN: 26 Jul 2005
Entered Medline: 25 Jul 2005

AB BRCA1 is a tumor suppressor gene that functions in DNA repair. Basal-like tumors are a distinctive subtype of breast cancer defined by gene expression profiles. Hereditary BRCA1 breast tumors and basal-like sporadic tumors have a similar phenotype and gene expression signature, suggesting involvement of BRCA1 in the pathogenesis of sporadic basal-like cancer. This study evaluates the role of BRCA1 in sporadic breast tumorigenesis. BRCA1 protein expression and promoter methylation are compared to tumor histopathology and gene expression profiles. We find BRCA1 protein expression correlates with tumor mitotic rate, consistent with normal cell-cycle regulation of the BRCA1 gene.

Methylation is found in 21% of tumors and is associated with lower BRCA1 protein, but not with specific pathologic features. Basal-like tumors, defined by hierarchical clustering of gene expression, have infrequent BRCA1 methylation and high levels of BRCA1 protein expression consistent with their high mitotic rate. Tumors with BRCA1 promoter methylation are present in all expression clusters; however, a subgroup of ER-positive high-grade tumors has a significantly greater number of BRCA1 methylated tumors. Absence of BRCA1 promoter methylation and high levels of BRCA1 expression in basal-like sporadic tumors suggest alternate explanations for the phenotypic similarities of these tumors to hereditary BRCA1 tumors.

L5 ANSWER 17 OF 29 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2005643979 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15846514
TITLE: Genomic and gene expression signature of the pre-invasive testicular carcinoma in situ.
AUTHOR: Almstrup Kristian; Ottesen Anne Marie; Sonne Si Brask; Hoei-Hansen Christina E; Leffers Henrik; Rajpert-De Meyts Ewa; Skakkebaek Niels E
CORPORATE SOURCE: University Department of Growth and Reproduction, Section GR-5064, Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark.. kristian@almstrup.net
SOURCE: Cell and tissue research, (2005 Oct) Vol. 322, No. 1, pp. 159-65. Electronic Publication: 2005-11-03. Ref: 45
Journal code: 0417625. ISSN: 0302-766X.
PUB. COUNTRY: Germany: Germany, Federal Republic of
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200602
ENTRY DATE: Entered STN: 6 Dec 2005
Last Updated on STN: 3 Feb 2006
Entered Medline: 3 Feb 2006

AB Testicular cancer is the most common malignancy among men in the reproductive age and the incidence is increasing, probably caused by environmental factors. Most testicular cancers are testicular germ cell tumours and all originate from a carcinoma in situ (CIS) pattern. In this review, we focus on the pre-invasive CIS and its possible fetal origin by reviewing recent data originating from DNA microarrays and comparative genomic hybridisations. A comparison of gene expression and genomic aberrations reveal chromosomal "hot spots" with mutual clustering of gene expression and genomic amplification. Some of the genes found in the hot spots may be involved in creating the CIS phenotype. On the other hand, many genes that are highly expressed in CIS are not present in the hot-spot areas. The gene expression profile of CIS thus most likely reflects the combined result of genomic amplification and increased transcriptional activation and/or deficiency in the epigenetic silencing of specific loci. Amplification of chromosome 12p, appears to be a good genomic marker of the transition from the pre-malignant to malignant CIS cell; this is consistent with recent findings of propagation advantages in cultured undifferentiated embryonic stem cells after spontaneous amplification in similar regions. The gene expression profile of CIS cells has remarkable similarity to that of embryonic stem cells and supports our long-standing hypothesis of an early developmental origin of CIS and testicular germ cell cancer.

L5 ANSWER 18 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2006:180377 BIOSIS
DOCUMENT NUMBER: PREV200600182489

TITLE: Characterization of distinct molecular signatures in myeloproliferative diseases with the JAK2V617F mutation and wild type JAK2.

AUTHOR(S): Ebert, Benjamin L. [Reprint Author]; Levine, Ross L.; Wadleigh, Martha; Brunet, Jean-Philippe; Pretz, Jennifer L.; Buque, Lambert; Lee, Stephanie J.; Gilliland, D. Gary; Golub, Todd R.

CORPORATE SOURCE: Dana Farber Canc Inst, Dept Med, Boston, MA 02115 USA

SOURCE: Blood, (NOV 16 2005) Vol. 106, No. 11, Part 1, pp. 39A. Meeting Info.: 47th Annual Meeting of the American-Society-of-Hematology. Atlanta, GA, USA. December 10 -13, 2005. Amer Soc Hematol.
CODEN: BLOOAW. ISSN: 0006-4971.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

ENTRY DATE: Entered STN: 15 Mar 2006
Last Updated on STN: 15 Mar 2006

AB The recently discovered JAK2V617F mutation provides a critical insight into the molecular pathogenesis of polycythemia vera and other myeloproliferative diseases (MPD). However, the mutations present in patients with wild type JAK2 have not been discovered, and the precise molecular consequences of JAK2 mutation have not been elucidated. We employed gene expression profiling to characterize the molecular phenotype of cells with the JAK2V617F mutation, to identify a distinct signature in cells without JAK2 mutations, and to refine a molecular taxonomy of MPDs. Using purified neutrophils from 70 patients with myeloproliferative diseases and 11 unaffected individuals, we performed gene expression profiling using oligonucleotide microarrays, sequencing of the JAK2 gene, quantitative genotyping by mass spectrometry and allele-specific quantitative PCR, and X-inactivation clonality assays. To reduce the confounding influence of normal neutrophils that are admixed with cells bearing disease-causing mutations, we examined the gene expression profiles of samples in which greater than 80% of JAK2 alleles bear the V617F mutation. PRV1, a previously identified marker of polycythemia vera, was powerfully overexpressed in neutrophils with a homozygous JAK2 mutation. In addition, cells with the JAK2 mutation had increased expression of a set of kinases, including JAK2, and decreased expression of a set of phosphatases. Cells that rely on JAK2 activation for clonal dominance may therefore derive a selective advantage from increased expression of the JAK2 gene. We next examined samples that have high clonality, and therefore relatively few normal neutrophils, but do not have a mutation in the JAK2 gene. These samples have a markedly different gene expression profile and overexpress a distinct set of kinases. The kinases overexpressed in cells with wild type JAK2 are candidates for further mutational analysis and are potential therapeutic targets. Utilizing these signatures and unsupervised analytical algorithms, the samples cluster according to their mutational status and the percentage of normal neutrophils in the sample. Our data demonstrate that the gene expression profiles of MPD samples are not uniform regardless of JAK2 genotype, implying that samples with and without JAK2 mutations have not activated the same pathway via alternative mechanisms. Moreover, we have identified a common signature in samples without JAK2 mutations that meets significance, indicating that one or a small number of mutations may play a critical role in these cells. Genotype and gene expression analyses are defining a molecular classification of myeloproliferative diseases with subtypes that have distinct therapeutic targets.

L5 ANSWER 19 OF 29 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2006288511 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 16622521

TITLE: Microarray analyses of peripheral blood cells identifies unique gene expression signature in psoriatic arthritis.

AUTHOR: Batliwalla Franak M; Li Wentian; Ritchlin Christopher T; Xiao Xiangli; Brenner Max; Laragione Teresina; Shao Tianmeng; Durham Robert; Kemshetti Sunil; Schwarz Edward; Coe Rodney; Kern Marlena; Baechler Emily C; Behrens Timothy W; Gregersen Peter K; Gulko Percio S

CORPORATE SOURCE: Robert S. Boas Center for Genomics and Human Genetics, Feinstein Institute for Medical Research, Manhasset, NY 11030, USA.

CONTRACT NUMBER: NO1-AR-1-2256 (NIAMS)

SOURCE: Molecular medicine (Cambridge, Mass.), (2005 Jan-Dec) Vol. 11, No. 1-12, pp. 21-9.
Journal code: 9501023. ISSN: 1076-1551.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE: Entered STN: 24 May 2006
Last Updated on STN: 26 May 2006

AB Psoriatic arthritis (PsA) is a chronic and erosive form of arthritis of unknown cause. We aimed to characterize the PsA phenotype using gene expression profiling and comparing it with healthy control subjects and patients rheumatoid arthritis (RA). Peripheral blood cells (PBCs) of 19 patients with active PsA and 19 age- and sex-matched control subjects were used in the analyses of PsA, with blood samples collected in PaxGene tubes. A significant alteration in the pattern of expression of 313 genes was noted in the PBCs of PsA patients on Affymetrix U133A arrays: 257 genes were expressed at reduced levels in PsA, and 56 genes were expressed at increased levels, compared with controls. Downregulated genes tended to cluster to certain chromosomal regions, including those containing the psoriasis susceptibility loci PSORS1 and PSORS2. Among the genes with the most significantly reduced expression were those involved in downregulation or suppression of innate and acquired immune responses, such as SIGIRR, STAT3, SHP1, IKBKB, IL-11RA, and TCF7, suggesting inappropriate control that favors proin-flammatory responses. Several members of the MAPK signaling pathway and tumor suppressor genes showed reduced expression. Three proinflammatory genes--S100A8, S100A12, and thioredoxin--showed increased expression. Logistic regression and recursive partitioning analysis determined that one gene, nucleoporin 62 kDa, could correctly classify all controls and 94.7% of the PsA patients. Using a dataset of 48 RA samples for comparison, the combination of two genes, MAP3K3 followed by CACNA1S, was enough to correctly classify all RA and PsA patients. Thus, PBC gene expression profiling identified a gene expression signature that differentiated PsA from RA, and PsA from controls. Several novel genes were differentially expressed in PsA and may prove to be diagnostic biomarkers or serve as new targets for the development of therapies.

L5 ANSWER 20 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 8

ACCESSION NUMBER: 2004:399112 BIOSIS

DOCUMENT NUMBER: PREV200400395658

TITLE: Different gene expression profiles between microsatellite instability-high and microsatellite stable colorectal carcinomas.

AUTHOR(S): Kim, Hyunki; Nam, Suk Woo; Rhee, Hwanseok; Li, Long Shan; Kang, Hyun Ju; Koh, Kwi Hye; Kim, Nam Kyu; Song, Jaehwi; Liu, Edison Tak-Bun; Kim, Hoguen [Reprint Author]

CORPORATE SOURCE: Coll MedDept Pathol, Yonsei Univ, CPO Box 8044, Seoul, 120752, South Korea

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SOURCE: Oncogene, (August 19 2004) Vol. 23, No. 37, pp. 6218-6225. print.
ISSN: 0950-9232 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 13 Oct 2004
Last Updated on STN: 13 Oct 2004

AB Recent molecular genetic studies have revealed that two major types of genomic instabilities, chromosomal instability (CIN) and microsatellite instability (MSI), exist in colorectal carcinomas. In order to clarify the molecular signature related to the CIN and MSI in colorectal carcinomas, we performed transcriptomic expression analysis on eight microsatellite instability-high (MSI-H) colorectal carcinomas and compared the results obtained with that of nine microsatellite stable (MSS) colorectal carcinomas using oligonucleotide microarrays containing 17 334 known genes and 1331 unknown genes or expression sequence tags ( ESTs). Unsupervised two-way hierarchical clustering with 5724 genes successfully classified tumors from normal mucosa, and displayed a distinctive MSI-H carcinomas subgroup. Based on intensive filtering, 57 known genes and eight ESTs were found to be highly relevant to the differentiation of MSI-H and MSS colorectal carcinomas. These genes successfully distinguish the new test set of six MSI-H and five MSS colorectal carcinomas. Many up- and downregulated genes in MSI-H colorectal carcinomas were related to the previously reported phenotypic characteristics; increased mucin production and intense peritumoral immune response in MSI-H carcinomas. Some of these differences were confirmed by semiquantitative reverse transcription-PCR and immunohistochemical analysis. Our findings indicate that there are many different genetic and transcriptomic characteristics between MSI-H and MSS colorectal carcinomas, and some of these differently expressed genes can be used as diagnostic or prognostic markers.

L5 ANSWER 21 OF 29 MEDLINE on STN
ACCESSION NUMBER: 2004060828 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14762059
TITLE: Comparative genomics of gene expression in the parasitic and free-living nematodes Strongyloides stercoralis and Caenorhabditis elegans.
AUTHOR: Mitreva Makedonka; McCarter James P; Martin John; Dante Mike; Wylie Todd; Chiapelli Brandi; Pape Deana; Clifton Sandra W; Nutman Thomas B; Waterston Robert H
CORPORATE SOURCE: Department of Genetics, Washington University School of Medicine, St Louis, Missouri 63108, USA..
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CONTRACT NUMBER: AI46593 (NIAID)
SOURCE: Genome research, (2004 Feb) Vol. 14, No. 2, pp. 209-20.
Journal code: 9518021. ISSN: 1088-9051.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AW495499; GENBANK-AW495500; GENBANK-AW495501; GENBANK-AW495502; GENBANK-AW495503; GENBANK-AW495504; GENBANK-AW495505; GENBANK-AW495506; GENBANK-AW495507; GENBANK-AW495508; GENBANK-AW495509; GENBANK-AW495510; GENBANK-AW495511; GENBANK-AW495512; GENBANK-AW495513; GENBANK-AW495514; GENBANK-AW495515; GENBANK-AW495516; GENBANK-AW495517; GENBANK-AW495518; GENBANK-AW495519; GENBANK-AW495520; GENBANK-AW495521; GENBANK-AW495522; GENBANK-AW495523; GENBANK-AW495524; GENBANK-AW495525; GENBANK-AW495526; GENBANK-AW495527; GENBANK-AW495528;

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AB Although developmental timing of gene expression is used to infer potential gene function, studies have yet to correlate this information between species. We analyzed 10,921 ESTs in 3311 clusters from first- and infective third-stage larva (L1, L3i) of the parasitic nematode Strongyloides stercoralis and compared the results to Caenorhabditis elegans, a species that has an L3i-like dauer stage. In the comparison of S. stercoralis clusters with stage-specific expression to C. elegans homologs expressed in either dauer or nondauer stages, matches between S. stercoralis L1 and C. elegans nondauer-expressed genes dominated, suggesting conservation in the repertoire of genes expressed during growth in nutrient-rich conditions. For example, S. stercoralis collagen transcripts were abundant in L1 but not L3i, a pattern consistent with C. elegans collagens. Although a greater proportion of S. stercoralis L3i than L1 genes have homologs among the C. elegans dauer-specific transcripts, we did not uncover evidence of a robust conserved L3i/dauer 'expression signature.' Strikingly, in comparisons of S. stercoralis clusters to C. elegans homologs with RNAi knockouts, those with significant L1-specific expression were more than twice as likely as L3i-specific clusters to match genes with phenotypes. We also provide functional classifications of S. stercoralis clusters.

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ACCESSION NUMBER: 2005:476729 BIOSIS

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TITLE: Gender counts: Defining the core genetic profile of polycythemia.

AUTHOR(S): Spivak, Jerry L. [Reprint Author]; Jie, Chunfa; Williams, Donna M.; Moliterno, Alison R.

CORPORATE SOURCE: Johns Hopkins Univ, Sch Med, Baltimore, MD USA

SOURCE: Blood, (NOV 16 2004) Vol. 104, No. 11, Part 1, pp. 190A. Meeting Info.: 46th Annual Meeting of the American-Society-of-Hematology. San Diego, CA, USA. December 04 -07, 2004. Amer Soc Hematol. CODEN: BLOOAW. ISSN: 0006-4971.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)

LANGUAGE: English

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AB Intra-abdominal venous thrombosis and exuberant extramedullary hematopoiesis leading to painful splenomegaly and hepatomegaly are two of the most serious consequences of polycythemia vera. Although polycythemia vera is slightly more common in men, both of these complications appear to

be more common in women, in whom the disease also presents at an earlier age. We previously reported that polycythemia vera peripheral blood (pb) CD34+ cells could be distinguished from their immunophenotypically similar normal counterparts by gene expression profiling. We, therefore, hypothesized that phenotypic differences in disease behavior between men and women with polycythemia vera were a consequence of gender-based genotypic differences and have employed gene expression profiling to examine this issue. For this purpose, cRNA was prepared from the total RNA of ph CD34+ cells of eleven polycythemia vera patients and six normal controls purified by immunomagnetic bead chromatography. The cRNA was hybridized to an Affymetrix HU133 high-density oligonucleotide microarray chip representing 22,000 genes. Approximately 30-45% of chip genes were recorded as present in the RNA samples. GC-RMA (Robust Multiarray Analysis) was used for normalization, to adjust for probe effects and for signal estimation. A parametric empirical Bayes statistical modeling method was used for differential gene expression analysis between the patients and controls. A posterior probability of >0.5 was taken to indicate significant differential gene expression. As previously reported, all polycythemia vera patients could be unequivocally distinguished from the controls, indicating that gene expression profiling can be employed as diagnostic test for polycythemia vera. With respect to gender, comparing male patients with male controls, 1106 genes were differentially expressed in the patient group: comparing female patients with female controls, 461 genes were differentially expressed in the patient group. Using unsupervised hierarchical clustering, the control patients segregated as a single group while the polycythemia vera patients segregated into two groups based on exuberant extramedullaryhematopoiesis but this segregation was not gender-based despite the marked differences in gender-specific gene expression. Importantly, however, after eliminating gender-specific genes, only 93 genes were concordantly expressed in the male and female patients (59 up regulated and 34 down regulated). This smallgroup appears to represent core genes in polycythemia vera whose behavior maybe modified by gender-specific genes but the specific up regulation or repression of which is essential for disease expression. These data indicate that gender-specific effects on gene expression must be addressed in order to determine the basic genetic signature of a hematologic malignancy and they also define a core genetic profile in polycythemia vera.

L5 ANSWER 23 OF 29 WPIDS COPYRIGHT 2006 THE THOMSON CORP on STN
ACCESSION NUMBER: 2003-493491 [46] WPIDS
DOC. NO. NON-CPI: N2003-391988
DOC. NO. CPI: C2003-132184
TITLE: Creating coherent data set to model biological systems, by storing data linked to unique identifier of biological sample into computer, converting linked data to numeric format that is converted to common unit system data.
DERWENT CLASS: B04 C07 D16 T01
INVENTOR(S): ALLEN, K; BEECHER, C; BOYES, D; COFFIN, M; DAVIS, K; HAMILTON, C; HOFFMAN, N; HURBAN, P; LAWRENCE, M; LIDDELL, C M; SHUSTER, J; WOESSNER, J; ZHANG, W; BROADWELL, D; GLASSBROOK, N; NYE, G J; POPA-BURKE, I; RANASINGHE, Y; WINFIELD, S; HURBAN, P M; LIDDELL, C; MULPURI, R; NYE, G; SLATER, T; TANZER, M
PATENT ASSIGNEE(S): (PARA-N) PARADIGM GENETICS INC; (ALLE-I) ALLEN K; (BEEC-I) BEECHER C; (BOYE-I) BOYES D; (COFF-I) COFFIN M; (DAVI-I) DAVIS K; (HAMI-I) HAMILTON C; (HOFF-I) HOFFMAN N; (HURB-I) HURBAN P; (LAWR-I) LAWRENCE M; (LIDD-I) LIDDELL C M; (SHUS-I) SHUSTER J; (WOES-I) WOESSNER J; (ZHAN-I) ZHANG W; (BROA-I) BROADWELL D; (GLAS-I) GLASSBROOK N; (NYEG-I) NYE G J; (POPA-I) POPA-BURKE I;

(RANA-I) RANASINGHE Y; (WINF-I) WINFIELD S; (HURB-I) HURBAN P M; (ICOR-N) ICORIA INC

COUNTRY COUNT: 101

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| | | Provisional | US 2001-344953P | 20011221 |
| | | Provisional | US 2002-356994P | 20020214 |
| | | Provisional | US 2002-363685P | 20020312 |
| | | Provisional | US 2002-368776P | 20020329 |
| | | Provisional | US 2002-372679P | 20020415 |
| | | Provisional | US 2002-374229P | 20020419 |
| | | Provisional | US 2002-379562P | 20020510 |
| | | Provisional | US 2002-384445P | 20020530 |
| | | Provisional | US 2002-404233P | 20020816 |
| | | Provisional | US 2002-407840P | 20020903 |
| | | Provisional | US 2002-408721P | 20020906 |
| | | Provisional | US 2002-414488P | 20020927 |
| | | | US 2002-300166 | 20021120 |
| US 2004019430 | A1 | Provisional | US 2001-331948P | 20011121 |
| | | Provisional | US 2001-344953P | 20011221 |
| | | Provisional | US 2002-356994P | 20020214 |
| | | Provisional | US 2002-363685P | 20020312 |
| | | Provisional | US 2002-368776P | 20020329 |
| | | Provisional | US 2002-372679P | 20020415 |
| | | Provisional | US 2002-374229P | 20020419 |
| | | Provisional | US 2002-379562P | 20020510 |
| | | Provisional | US 2002-384445P | 20020530 |
| | | Provisional | US 2002-404233P | 20020816 |
| | | Provisional | US 2002-407840P | 20020903 |
| | | Provisional | US 2002-408721P | 20020906 |
| | | Provisional | US 2002-414488P | 20020927 |
| | | | US 2002-300291 | 20021120 |
| US 2004019435 | A1 | Provisional | US 2001-331948P | 20011121 |
| | | Provisional | US 2001-344953P | 20011221 |
| | | Provisional | US 2002-356994P | 20020214 |
| | | Provisional | US 2002-363685P | 20020312 |
| | | Provisional | US 2002-368776P | 20020329 |
| | | Provisional | US 2002-372679P | 20020415 |
| | | Provisional | US 2002-374229P | 20020419 |
| | | Provisional | US 2002-379562P | 20020510 |
| | | Provisional | US 2002-384445P | 20020530 |
| | | Provisional | US 2002-404233P | 20020816 |
| | | Provisional | US 2002-407840P | 20020903 |
| | | Provisional | US 2002-408721P | 20020906 |
| | | Provisional | US 2002-414488P | 20020927 |
| | | | US 2002-300360 | 20021120 |
| AU 2002352831 | A1 | | AU 2002-352831 | 20021120 |
| EP 1481356 | A1 | | EP 2002-789787 | 20021120 |
| | | | WO 2002-US37301 | 20021120 |
| US 6873914 | B2 | Provisional | US 2001-331948P | 20011121 |
| | | Provisional | US 2001-344953P | 20011221 |
| | | Provisional | US 2002-356994P | 20020214 |
| | | Provisional | US 2002-363685P | 20020312 |
| | | Provisional | US 2002-368776P | 20020329 |
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| Provisional | US 2002-379562P | 20020510 |
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| Provisional | US 2002-404233P | 20020816 |
| Provisional | US 2002-407840P | 20020903 |
| Provisional | US 2002-408721P | 20020906 |
| Provisional | US 2002-414488P | 20020927 |
| | US 2002-300360 | 20021120 |

FILING DETAILS:

| PATENT NO | KIND | PATENT NO |
|---|---|---|
| AU 2002352831 | A1 Based on | WO 2003046798 |
| EP 1481356 | A1 Based on | WO 2003046798 |

PRIORITY APPLN. INFO: US 2002-414488P 20020927; US
2001-331948P 20011121; US
2001-344953P 20011221; US
2002-356994P 20020214; US
2002-363685P 20020312; US
2002-368776P 20020329; US
2002-372679P 20020415; US
2002-374229P 20020419; US
2002-379562P 20020510; US
2002-384445P 20020530; US
2002-404233P 20020816; US
2002-407840P 20020903; US
2002-408721P 20020906; US
2002-300262 20021120; US
2002-300204 20021120; US
2002-300599 20021120; US
2002-300598 20021120; US
2002-300184 20021120; US
2002-300543 20021120; US
2002-300551 20021120; US
2002-300166 20021120; US
2002-300291 20021120; US
2002-300360 20021120

AN 2003-493491 [46] WPIDS

AB WO2003046798 A UPAB: 20030719

NOVELTY - Creating coherent data set for modeling biological systems, comprising entering unique identifier of at least one biological sample into computer tracking system, storing data in computer tracking system, where the data are linked to unique identifier, converting linked data to a numeric format which is converted to a common unit system, where the system data are a coherent data set which serves as model for biological system, is new.

DETAILED DESCRIPTION - Creating (M1) coherent data set for modeling biological systems, comprising:

(a) entering unique identifier of at least one biological sample into computer tracking system;

(b) storing data in computer tracking system, where the data are linked to unique identifier;

(c) converting linked data to a numeric format; and

(d) converting numeric format data to common unit system, where the common unit system data are a coherent data set which serves as model for biological system.

The method additionally comprises the step of reducing the dimensionality of the common unit system data, where the dimensionally reduced common unit system data are a coherent data set which serves as a model for a biological system. (M1) can optionally be carried out in various formats as given in specification e.g. creating coherent data set

for modeling biological systems, involves carrying out steps (a), (b) and (c) as described in (M1), transforming the numeric format data into a Gaussian distribution, converting the Gaussian distribution data to a common unit system, and reducing the dimensionality of the common unit system data, where the dimensionally reduced common unit system data are a coherent data set which serves as a model for a biological system. The reducing step is optional, where the Gaussian distribution data is converted to a common unit system data which serves as model for biological system.

INDEPENDENT CLAIMS are also included for the following:

(1) a system for creating coherent data for modeling biological systems;

(2) establishing signature profile indicative of physiological status of an individual, comprising:

(a) entering unique identifier of at least one biological sample into computer tracking system;

(b) storing data in computer tracking system, where the data are linked to unique identifier;

(c) comparing linked data to a reference; and

(d) determining the most informative of the compared data, which is a signature profile indicative of physiological status;

(3) a system for establishing a signature profile indicative of physiological status of an individual;

(4) examining (M2) chemical components in a biological sample, comprising:

(a) entering a unique identifier of at least one biological sample into a computer tracking system;

(b) simultaneously collecting data from the sample, for several peaks, each peak comprising at least one chemical component, where the data comprise data from at least two processes, storing the data in the computer tracking system, where the data are linked to the unique identifier; and

(c) characterizing and/or identifying the chemical components; and

(5) a system for examining chemical components and metabolites in a biological sample.

USE - (M1) is useful for creating a coherent data set for modeling biological systems, where preferably the coherent data set is a model for a particular disease or disease stage, or a model for the efficacy of a therapeutic program or exposure to a particular chemical. (M1) is useful for establishing a signature profile indicative of physiological status of an individual which involves carrying out steps (a)-(d) of (M1) and then determining the most informative of the common unit system data, where the most informative data are a signature profile indicative of a physiological status. (M2) is useful for examining metabolites in a biological sample which involves carrying (M2) and linking the characterized and/or identified chemical components to metabolites in biochemical pathways. (All claimed.) (M1) is useful for creating coherent data sets which are useful for numerous biological applications, such as, for example, determining gene function, identifying and validating drug and pesticide targets, identifying and validating drug and pesticide candidate compounds, profiling of drug and pesticide compounds, producing a compilation of health or wellness profiles, determining compound site(s) of action, identifying unknown samples, and numerous other applications.

ADVANTAGE - The resolution power of coherent data sets is enormous, as not only can different types of data from a single organism be combined and directly compared for improved representation of an entire biological system or organism, but data from completely different organisms can be analyzed together in a coherent data set for similarities and differences. After new targets for drug, pesticide, and nutriceutical applications are identified, there remains a long and difficult process for the development of an effective aimed at the identified target. The methods maximize

efficiency in bringing targets to product development. By using data derived from multiple biological indicators of physiological status, compelling targets can be more thoroughly validated and optimized for greatest effectiveness. The methods and systems allow biological samples to be screened using multiple technologies, providing for the simultaneous examination of disparate indicators of biological status, so that the effect of a particular chemical compound on a sample can be understood more thoroughly than was historically possible. Creation of coherent data sets allows subtle and complex effects to be observed so that target and lead compound identification, validation selection are more efficient. The optimization of lead compounds is more efficient as well, as it is possible to optimize the application of the selected leads, and screen-out selected leads based on parameters such as toxicity. The methods and systems allow for the development of highly efficacious products while spending as little time and money as possible at a discovery stage. The methods lower the cost of drug discovery, decrease the time to market for new drugs, lower the incidence of adverse toxic side effects, and complement other genomics tools to help researchers better understand the link between cellular or biochemical function, pharmaceutical compounds, toxicity, and disease response.
Dwg.0/23

L5 ANSWER 24 OF 29 SCISEARCH COPYRIGHT (c) 2006 The Thomson Corporation on STN

ACCESSION NUMBER: 2003:1042282 SCISEARCH
THE GENUINE ARTICLE: 743LR
TITLE: Gene expression patterns in human embryonic stem cells and human pluripotent germ cell tumors
AUTHOR: Sperger J M; Chen X; Draper J S; Antosiewicz J E; Chon C H; Jones S B; Brooks J D; Andrews P W; Brown P O (Reprint); Thomson J A
CORPORATE SOURCE: 1220 Capitol Court, Madison, WI 53715 USA (Reprint); Univ Wisconsin, Sch Med, Wisconsin Natl Primate Res Ctr, Madison, WI 53715 USA; Univ Wisconsin, Sch Med, Dept Anat, Madison, WI 53715 USA; Stanford Univ, Sch Med, Howard Hughes Med Inst, Dept Urol, Stanford, CA 94305 USA; Stanford Univ, Sch Med, Howard Hughes Med Inst, Dept Biochem, Stanford, CA 94305 USA; Univ Sheffield, Dept Biomed Sci, Sheffield S10 27N, S Yorkshire, England
COUNTRY OF AUTHOR: USA; England
SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA, (11 NOV 2003) Vol. 100, No. 23, pp. 13350-13355.
ISSN: 0027-8424.
PUBLISHER: NATL ACAD SCIENCES, 2101 CONSTITUTION AVE NW, WASHINGTON, DC 20418 USA.
DOCUMENT TYPE: Article; Journal
LANGUAGE: English
REFERENCE COUNT: 55
ENTRY DATE: Entered STN: 9 Dec 2003
Last Updated on STN: 9 Dec 2003

*ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS*

AB Remarkably little is known about the transcriptional profiles of human embryonic stem (ES) cells or the molecular mechanisms that underlie their pluripotency. To identify commonalties among the transcriptional profiles of different human pluripotent cells and to search for clues into the genesis of human germ cell tumors, we compared the expression profiles of human ES cell lines, human germ cell tumor cell lines and tumor samples, somatic cell lines, and testicular tissue samples by using cDNA microarray analysis. Hierarchical cluster analysis of gene expression profiles showed that the five independent human ES cell lines clustered tightly together, reflecting highly similar expression

profiles. The gene expression patterns of human ES cell lines showed many similarities with the human embryonal carcinoma cell samples and more distantly with the seminoma samples. We identified 895 genes that were expressed at significantly greater levels in human ES and embryonal carcinoma cell lines than in control samples. These genes are candidates for involvement in the maintenance of a pluripotent, undifferentiated phenotype.

L5 ANSWER 25 OF 29 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2003195104 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12713594
TITLE: Gene expression profiles of cutaneous B cell lymphoma.
AUTHOR: Storz Monique N; van de Rijn Matt; Kim Youn H; Mraz-Gernhard Serena; Hoppe Richard T; Kohler Sabine
CORPORATE SOURCE: Department of Pathology, Division of Medical Oncology, Stanford University Medical Center, Stanford, California 94305, USA.
SOURCE: The Journal of investigative dermatology, (2003 May) Vol. 120, No. 5, pp. 865-70.
Journal code: 0426720. ISSN: 0022-202X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200306
ENTRY DATE: Entered STN: 26 Apr 2003
Last Updated on STN: 6 Jun 2003
Entered Medline: 5 Jun 2003

AB We studied gene expression profiles of 17 cutaneous B cell lymphomas that were collected with 4-6 mm skin punch biopsies. We also included tissue from two cases of mycosis fungoides, three normal skin biopsies, and three tonsils to create a framework for further interpretation. A hierarchical cluster algorithm was applied for data analysis. Our results indicate that small amounts of skin tissue can be used successfully to perform microarray analysis and result in distinct gene expression patterns. Duplicate specimens clustered together demonstrating a reproducible technique. Within the cutaneous B cell lymphoma specimens two specific B cell differentiation stage signatures of germinal center B cells and plasma cells could be identified. Primary cutaneous follicular and primary cutaneous diffuse large B cell lymphomas had a germinal center B cell signature, whereas a subset of marginal zone lymphomas demonstrated a plasma cell signature. Primary and secondary follicular B cell lymphoma of the skin were closely related, despite previously reported genetic and phenotypic differences. In contrast primary and secondary cutaneous diffuse large B cell lymphoma were less related to each other. This pilot study allows a first glance into the complex and unique microenvironment of B cell lymphomas of the skin and provides a basis for future studies, which may lead to the identification of potential histologic and prognostic markers as well as therapeutic targets.

L5 ANSWER 26 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN
ACCESSION NUMBER: 2003:530219 BIOSIS
DOCUMENT NUMBER: PREV200300525932
TITLE: A COMPARATIVE MICROARRAY ANALYSIS OF MORPHOLOGICALLY NORMAL RPE CELLS LASER CAPTURED MICRODISSECTED FROM THE MACULA AND PERIPHERY OF HUMAN DONOR GLOBES.
AUTHOR(S): Ishibashi, K. [Reprint Author]; Tian, J. [Reprint Author]; Handa, J. [Reprint Author]
CORPORATE SOURCE: Ophthalmology, Johns Hopkins University, Baltimore, MD, USA

SOURCE: ARVO Annual Meeting Abstract Search and Program Planner, (2003) Vol. 2003, pp. Abstract No. 2292. cd-rom. Meeting Info.: Annual Meeting of the Association for Research in Vision and Ophthalmology. Fort Lauderdale, FL, USA. May 04-08, 2003. Association for Research in Vision and Ophthalmology.
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)
LANGUAGE: English
ENTRY DATE: Entered STN: 12 Nov 2003
Last Updated on STN: 12 Nov 2003

AB Purpose: To determine the expression profiles of morphologically normal RPE cells overlying nonthickened Bruchs membrane from the macula and periphery of human donor globes. Methods: Morphologically normal RPE cells overlying nonthickened Bruchs membrane (n=5000 cells) from 10 donor eyes (ages 71-83 years) were laser capture microdissected. The RNA was extracted by column purification, and first strand cDNA probes were amplified using Super SMART. Probes were radiolabeled with 33P-dATP and hybridized to ResGens known human membrane array (n=4325 genes). The signal was visualized with phosphorimager analysis. The TIFF image was analyzed by ResGens Pathways 3 software. The data were normalized to a mean signal reference array, and analyzed using Cluster/Treeview and SAM. Real time PCR was used to confirm differential expression of selected genes. Results: An average of 95% of genes on the array were expressed. 114 of 165 genes (69%) were found expressed by macular laser captured RPE cells on this array that were identified from a recent RPE specific library. Cluster analysis in 6 of 10 eyes showed that the expression signature of macular and peripheral cells clustered by donor. No significant clustering by topographical origin of RPE cells was identified. Using SAM analysis with a 10% false discovery rate, 10 genes were identified as differentially expressed between macular and peripheral RPE. These genes were related to cell cycle regulation (n=5), metabolism (n=3), cell differentiation (n=1), and apoptosis (n=1). Conclusions: Microarray analysis of this set of globes suggests that the gene expression profile between normal macular and peripheral RPE cells is similar and most related to the specific donor. Differences in genes important to the general maintenance of the cell may influence phenotypic differences between macular and peripheral cells.

L5 ANSWER 27 OF 29 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2002374719 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12119265
TITLE: Dissecting the circuitry of protein kinase A and cAMP signaling in cancer genesis: antisense, microarray , gene overexpression, and transcription factor decoy.
AUTHOR: Cho-Chung Yoon S; Nesterova Maria; Becker Kevin G; Srivastava Rakesh; Park Yun Gyu; Lee Youl Nam; Cho Yee Sook; Kim Meyoung-Kin; Neary Catherine; Cheadle Chris
CORPORATE SOURCE: Cellular Biochemistry Section, BRL, CCR, National Cancer Institute, National Institutes of Health, Bethesda, Maryland 20892-1750, USA.. yc12b@nih.gov
CONTRACT NUMBER: N02-BC-76212/C270012 (NCI)
SOURCE: Annals of the New York Academy of Sciences, (2002 Jun) Vol. 968, pp. 22-36. Ref: 86
Journal code: 7506858. ISSN: 0077-8923.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 200208
ENTRY DATE: Entered STN: 18 Jul 2002
Last Updated on STN: 21 Aug 2002
Entered Medline: 20 Aug 2002

AB Expression of the RI alpha subunit of the cAMP-dependent protein kinase type I (PKA-I) is enhanced in human cancer cell lines, in primary tumors, in transformed cells, and in cells upon stimulation of growth. Signaling via the cAMP pathway may be complex, and the biological effects of the pathway in normal cells may depend upon the physiological state of the cells. However, results of different experimental approaches such as antisense exposure, 8-Cl-cAMP treatment, and gene overexpression have shown that the inhibition of RI alpha/PKA-I exerts antitumor activity in a wide variety of tumor-derived cell lines examined in vitro and in vivo. cDNA microarrays have further shown that in a sequence-specific manner, RI alpha antisense induces alterations in the gene expression profile of cancer cells and tumors. The cluster of genes that define the "proliferation-transformation" signature are down-regulated, and those that define the "differentiation-reverse transformation" signature are up-regulated in antisense-treated cancer cells and tumors, but not in host livers, exhibiting the molecular portrait of the reverted (flat) phenotype of tumor cells. These results reveal a remarkable cellular regulation, elicited by the antisense RI alpha, superimposed on the regulation arising from the Watson-Crick base-pairing mechanism of action. Importantly, the blockade of both the PKA and PKC signaling pathways achieved with the CRE-transcription factor decoy inhibits tumor cell growth without harming normal cell growth. Thus, a complex circuitry of cAMP signaling comprises cAMP growth regulatory function, and deregulation of the effector molecule by this circuitry may underlie cancer genesis and tumor progression.

L5 ANSWER 28 OF 29 MEDLINE on STN
ACCESSION NUMBER: 1999232505 MEDLINE
DOCUMENT NUMBER: PubMed ID: 10216859
TITLE: Identification of Yersinia enterocolitica genes affecting survival in an animal host using signature-tagged transposon mutagenesis.
AUTHOR: Darwin A J; Miller V L
CORPORATE SOURCE: Department of Molecular Microbiology, Washington University School of Medicine, St Louis, MO 63110, USA.
CONTRACT NUMBER: AI01230 (NIAID)
SOURCE: Molecular microbiology, (1999 Apr) Vol. 32, No. 1, pp. 51-62.
Journal code: 8712028. ISSN: 0950-382X.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 15 Jul 1999
Last Updated on STN: 15 Jul 1999
Entered Medline: 7 Jul 1999

AB Pathogenic Yersinia species are associated with both localized and systemic infections in mammalian hosts. In this study, signature -tagged transposon mutagenesis was used to identify Yersinia enterocolitica genes required for survival in a mouse model of infection. Approximately 2000 transposon insertion mutants were screened for attenuation. This led to the identification of 55 mutants defective for survival in the animal host, as judged by their ability to compete with the wild-type strain in mixed infections. A total of 28 mutants had transposon insertions in the virulence plasmid, validating the screen. Two of the plasmid mutants with severe virulence defects had insertions in an uncharacterized region. Several of the chromosomal

insertions were in a gene cluster involved in O-antigen biosynthesis. Other chromosomal insertions identified genes not previously demonstrated as being required for in vivo survival of Y. enterocolitica. These include genes involved in the synthesis of outer membrane components, stress response and nutrient acquisition. One severely attenuated mutant had an insertion in a homologue of the pspC gene (phage shock protein C) of Escherichia coli. The phage shock protein operon has no known biochemical or physiological function in E. coli, but is apparently essential for the survival of Y. enterocolitica during infection.

L5 ANSWER 29 OF 29 BIOSIS COPYRIGHT (c) 2006 The Thomson Corporation on STN DUPLICATE 11

ACCESSION NUMBER: 1997:220627 BIOSIS
DOCUMENT NUMBER: PREV199799512343
TITLE: Proposal for a new hierarchic classification system, Actinobacteria classis nov.
AUTHOR(S): Stackebrandt, Erko [Reprint author]; Rainey, Fred A.; Ward-Rainey, Naomi L.
CORPORATE SOURCE: DSMZ Deutsche Sammlung Mikroorganismen Zellkulturen GmbH, Mascheroder Weg 1b, 38124 Braunschweig, Germany
SOURCE: International Journal of Systematic Bacteriology, (1997) Vol. 47, No. 2, pp. 479-491.
CODEN: IJSBA8. ISSN: 0020-7713.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 May 1997
Last Updated on STN: 22 May 1997

AB A new hierarchic classification structure for the taxa between the taxonomic levels of genus and class is proposed for the actinomycete line of descent as defined by analysis of small subunit (16S) rRNA and genes coding for this molecule (rDNA). While the traditional circumscription of a genus of the actinomycete subphylum is by and large in accord with the 16S rRNA/rDNA-based phylogenetic clustering of these organisms, most of the higher taxa proposed in the past do not take into account the phylogenetic clustering of genera. The rich chemical, morphological and physiological diversity of phylogenetically closely related genera makes the description of families and higher taxa so broad that they become meaningless for the description of the enclosed taxa. Here we present a classification system in which phylogenetically neighboring taxa at the genus level are clustered into families, suborders, orders, subclasses, and a class irrespective of those phenotypic characteristics on which the delineation of taxa has been based in the past. Rather than being based on a listing of a wide array of chemotaxonomic, morphological, and physiological properties, the delineation is based solely on 16S rDNA/rRNA sequence-based phylogenetic clustering and the presence of taxon-specific 16S rDNA/RNA signature nucleotides.

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| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 118.51 | 118.93 |

| DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| CA SUBSCRIBER PRICE | -4.50 | -4.50 |

FILE 'STNGUIDE' ENTERED AT 16:05:26 ON 01 OCT 2006

AND TECHNOLOGY CORPORATION, AND FACHINFORMATIONSZENTRUM KARLSRUHE

FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Sep 29, 2006 (20060929/UP).

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(FILE 'HOME' ENTERED AT 15:56:56 ON 01 OCT 2006)

FILE 'MEDLINE, BIOSIS, CAPLUS, SCISEARCH, EMBASE, WPIDS' ENTERED AT
15:57:55 ON 01 OCT 2006
E MINOR JAMES/AU
E MINOR JAMES M/AU
L1 23 E3
L2 3 (CLUSTER OR CLUSTERING) AND L1
L3 3 DUP REM L2 (0 DUPLICATES REMOVED)
L4 59 (CLUSTER OR CLUSTERING) AND (PHENOTYPE OR PHENOTYPIC) AND SIGNA
L5 29 DUP REM L4 (30 DUPLICATES REMOVED)
L6 0 (INVERT OR INVERTING) AND L5

FILE 'STNGUIDE' ENTERED AT 16:05:26 ON 01 OCT 2006

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---Logging off of STN---

=>
Executing the logoff script...

=> LOG Y

| COST IN U.S. DOLLARS | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| FULL ESTIMATED COST | 2.70 | 121.63 |

| DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS) | SINCE FILE ENTRY | TOTAL SESSION |
|---|---|---|
| CA SUBSCRIBER PRICE | 0.00 | -4.50 |

STN INTERNATIONAL LOGOFF AT 16:32:24 ON 01 OCT 2006